

ABSTRACT

THESIS: *Staphylococcus aureus* stimulates the release of constitutive tissue factor in lung epithelial cells

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PAGES: 50

Sepsis is a life threatening condition caused by infectious agents, including the Gram-positive bacterium *Staphylococcus aureus*. Symptoms of sepsis often include intravascular coagulation and organ failure. Tissue factor (TF), the initiator of coagulation, may contribute to fibrin deposition in the lungs of patients with sepsis. We

have found that lung epithelial cells constitutively express TF on the cell surface and in intracellular pools. Levels of TF diminished in response to *S. aureus* invasion possibly indicating a release in the form of shedding vesicles. TF levels diminish in response to viable bacteria, but not in response to heat killed (HK) bacteria. Our studies indicate that bacterial attachment at the host cell surface is insufficient to diminish levels of constitutive TF. Finally, we established that levels of constitutive intracellular TF diminish in response to the bacterial toxin, α -hemolysin, alone. This approach may provide a basis for understanding the role of TF in coagulation seen in sepsis.